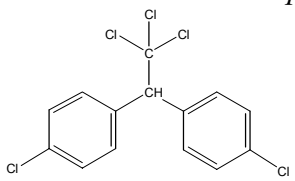
DDT (Dichlorodiphenyltrichloroethane; 1,1,1-Trichloro-2,2-bis(*p*-chlorophenyl)ethane)
CAS No. 50-29-3

First Listed in the *Fourth Annual Report on Carcinogens*



CARCINOGENICITY

DDT (dichlorodiphenyltrichloroethane) is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity in experimental animals (IARC S.4, 1982; IARC V.5, 1974). When administered orally in the diet or by stomach tube, DDT induced hepatomas in mice and rats of both sexes and lymphomas and lung carcinomas and adenomas in mice. When administered by subcutaneous injection, DDT induced liver tumors in mice of both sexes. The evidence for carcinogenicity of DDT administered orally was negative in studies with hamsters and inconclusive in studies with dogs and monkeys (IARC S.4, 1982; IARC V.5, 1974). The results of oral administration of TDE and DDE, structurally related to DDT and present as contaminants of technical-grade DDT, were conclusive. In male rats, TDE induced follicular cell carcinomas and adenomas of the thyroid. DDE induced hepatocellular carcinomas in mice of both sexes. Administration of technical grade DDT, TDE, and *p*,*p*′-DDE in the diet provided no evidence for the carcinogenicity of DDT in mice and rats (NCI 131, 1978).

There is inadequate evidence for the carcinogenicity of DDT in humans (IARC S.4, 1982). Four studies of cancer patients reported that these individuals had higher tissue levels of DDT than individuals dying from other causes. Other studies reporting elevated serum levels of DDT provided inadequate evidence for carcinogenicity (IARC S.4, 1982; IARC V.5, 1978).

PROPERTIES

DDT is the common name of the technical product consisting of 65%-80% *p*,*p*′-DDT. Technical DDT is a waxy solid that is odorless or may have a slight aromatic odor. Major impurities include *o,p*′-DDT (15%-21%), *p*,*p*′-TDE (up to 4%), 1-(*p*-chlorophenyl)-2,2,2-trichloroethanol (up to 1.5%), and traces of *o*,*o*′-DDT and bis(*p*-chlorophenyl) sulfone. Up to 1% *m*,*p*′-DDT may be present in some technical DDT.

p,*p*′-DDT occurs as colorless crystals with a fruit-like odor. It is very soluble in fats and most organic solvents and practically insoluble in water. *p*,*p*′-DDT can dehydrochlorinate at temperatures above its melting point and in organic solvents in the presence of alkalies or organic bases. *p*,*p*′-DDT is stable in strong acids and can withstand acid permanganate oxidation.

o,*p*′-DDT occurs as a crystalline solid which is soluble in fats and most organic solvents and is slightly soluble in water at 25 ^{o}C.

USE

DDT is currently used only under Public Health Service supervision as an insecticide in the United States for public health emergencies and by the USDA or military for health quarantine. EPA banned use of DDT in food in 1972 and in non food use in 1988 (EPA, 1998). In the United Kingdom it is used in the formulation of prescription drugs for the treatment of pediculosis (head lice, body lice, and crab lice). Formerly, DDT was used for the control of hundreds of insect pests in orchards, gardens, fields, and forests. In the public health field, DDT was used as a mosquito larvicide, as a residual spray for the eradication of malaria in dwellings, and as a dust in mass human delousing programs for typhus control. DDT has also been used for mothproofing clothing and on animals for spot treatment of screw worm-infested wounds (IARC V.5, 1974; HSDB, 1997).

PRODUCTION

Currently, no U.S. companies report the production of DDT, but major producers of the chemical exist outside the country (ATSDR, 1994-R035; SRIa, 1997). In 1985, two U.S. producers exported 666,600 lb of DDT (ATSDR, 1994-R035). The U.S. Bureau of the Census reported that an unpublished quantity of DDT was imported in 1985 (USDOC Imports, 1986). The 1979 TSCA Inventory identified two producers of DDT in 1977, but no production volume was provided. The CBI Aggregate was between 1 million and 100 million lb (TSCA, 1979). In 1972, U.S. imports of DDT totaled 440,000 lb and exports totaled 35 million lb. In 1971, U.S. production of DDT was 44 million lb. In 1963, U.S. production peaked at 188 million lb, with domestic consumption accounting for 61 million lb (NIOSH Review, 1978). Technical DDT was first synthesized in 1874, and by 1943 commercial production had begun (IARC V.5, 1975).

EXPOSURE

The primary routes of potential human exposure to DDT are inhalation, ingestion, and dermal contact. Studies, however, have shown that even with high doses minimal absorption of DDT through skin occurs. This pathway is therefore considered negligible. Since DDT is present in very low concentrations in the atmosphere, exposure through inhalation, too, is negligible (ATSDR, 1994-R035). Potential human exposure, nevertheless, is presumed to be widespread. Before 1945, all of the DDT produced in the United States was used by the military. When it became available commercially in 1945, the results were so spectacular that U.S. consumption reached 57 million lb in 1950. Domestic consumption peaked in 1959 at 78 million lb and declined to approximately 22 million lb in 1972. In June 1972, EPA issued a ban on all but minor uses of DDT, limiting its use in the United States to nearly zero (NIOSH Review, 1978). It has been estimated that 1.2 billion lb of DDT were used in the United States in the past (Chem. Eng. News, 1988a). Several studies have been carried out on the possible exposure of workers involved in the manufacture, formulation, and application of DDT. Potential dermal exposure estimates were 84 mg/hr for outdoor spraying, 1,775 mg/hr for indoor spraying, 212 mg/hr during forest spraying, and 524.5 mg/hr for formulating plant workers. Potential respiratory exposure estimates were 0.11 mg/hr for outdoor spraying, 7.1 mg/hr for indoor spraying, 4.9 mg/hr for forest spraying, and 14.1 mg/hr for formulating plant workers. In 1967, the daily average intake of DDT by 20 men with high occupational exposure was estimated to be 17.5 to 18 mg/man per day, as compared with an average of 0.04 mg/man per day for the general population (IARC V.5, 1974). The ACGIH (1986) recommended threshold limit value (TLV) is 1 mg/m^3 with no short-term exposure limit (STEL).

In spite of the 1972 U.S. ban of DDT, human exposure to DDT potentially is great because of its extensive former use and the persistence of the compound and its metabolites in the environment, but few persons should be exposed to high concentrations. DDT has been detected in air, rain, soil, water, animal and plant tissues, food and the work environment. Potential consumer exposure occurred from contact with mothproofing products and ingestion of fruits treated with DDT-containing pesticides. People living in the vicinity of fields and forests fumigated with these pesticides were potentially exposed to DDT dust and vapor. Today, exposure to DDT and its metabolites is primarily from ingestion of small amounts in the diet, particularly from meat, fish, poultry, and root and leafy vegetables. The residue levels have declined and continue to decline, but because of the persistence of the chemical, low levels will be present for decades (ATSDR, 1994-R035). In the recent Nonoccupational Pesticide Exposure Study (NOPES), DDT was found in house dust in five out of eight homes, which thus introduced the possible source of risk for toddlers through ingestion of the dust. The DDT is assumed to be tracked into the home on people's shoes from outdoor soil (Wallace, 1991). DDT also accumulates in fatty tissues (Farm Chemicals Handbook, 1985) and is therefore found in higher concentrations in human milk than in cow milk or other infant foods (ATSDR, 1994-R035).

Although the United States and other developed countries have banned the use of DDT, developing countries continue to use it because of its low cost of manufacture, its effectiveness, and its ease of application. In a study measuring concentrations of DDT metabolites in adipose tissue to assess chronic exposure in workers who control malaria vectors, levels were found to be six times greater than those of the general population. These were the result of multiple exposure routes, with inhalation being the major pathway (Rivero-Rodriguez et al., 1997).

REGULATIONS

DDT is regulated by EPA under the Clean Water Act (CWA), Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), Food, Drug, and Cosmetic Act (FD&CA), Resource Conservation and Recovery Act (RCRA), and Superfund Amendments and Reauthorization Act (SARA). Effluent discharge guidelines and water quality criteria have been set under CWA. DDT is subject to reporting rules under CWA and SARA. A reportable quantity (RQ) of 1 lb has been established under CERCLA and CWA. It is regulated as a hazardous constituent of waste under RCRA. Tolerances for residues of DDT in or on raw agricultural commodities have been established under FD&CA. In 1972, EPA canceled most DDT registrations under FIFRA. NIOSH has recommended that DDT exposure be limited to 0.5 mg/m^3 as a 10-hr TWA. OSHA has established a permissible exposure limit (PEL) of 1 mg/m^3 as an 8-hr time-weighted average (TWA). OSHA also regulates DDT under the Hazard Communication Standard and as a chemical hazard in laboratories. Regulations are summarized in Volume II, Table B-34.